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Borderline personality disorder traits and affect reactivity to positive affect induction followed by a stressor

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ABSTRACT

Background and objectives: Affective hyperreactivity is a core feature of Borderline Personality Disorder (BPD), yet little is known about reactivity of positive affect (PA). Objectives were to explore the relationship between BPD traits and affect reactivity in response to a personalized PA-induction and a subsequent stressor. Patient status (seeking outpatient treatment for personality-related problems; yes/no), depressive symptoms, and age were examined as alternative predictors of affect reactivity.

Methods: One hundred and eight females (35 patients) reported on their BPD and depressive symptoms. They completed the Best Possible Self-exercise and a modified Trier Social Stress Task. Trajectories of high and low arousal PA (HAP and LAP) and negative affect (NA) were analyzed with mixed regression modelling.

Results: Patient status (for HAP) and depressive symptoms (for LAP and NA) predicted affect reactivity better than BPD traits. Patients showed a weaker HAP increase after PA-induction, and a similar HAP decrease after the stressor, compared to non-patients. Higher depressive symptoms predicted stronger improvement of LAP and NA after PA-induction, and less pronounced deterioration of LAP and NA after the stressor, relative to baseline.

Limitations: The sample was a convenience sample amplified with outpatients. Future research should (1) use clinical groups, (2) randomize to neutral vs. PA-induction, and (3) continue to differentiate between HAP and LAP.

Conclusions: Our results do not support models postulating BPD-specific affective hyperreactivity. HAP and LAP have different trajectories, depending on the degree of psychopathology. The resilience-enhancing potential of a PA-focus in psychotherapy needs further research.

1. Introduction

Emotion dysregulation is a core feature of Borderline Personality Disorder (BPD). Linehan's biosocial theory posits a broad dysregulation of emotions, characterized by heightened emotional sensitivity and heightened emotion reactivity (Linehan, 1993). This broad dysregulation includes positive emotions: "The dysfunction proposed by Linehan is one of broad dysregulation across all aspects of emotional responding" (Crowell, Beauchaine, & Linehan, 2009, p.2).

Reactivity to unpleasant stimuli, especially in terms of negative affect reactivity, is reasonably well researched. Laboratory studies provide evidence for higher NA[1] reactivity to borderline-specific negative stimuli (e.g., related to abandonment or childhood abuse) or to personally-relevant unpleasant sounds in BPD (e.g., Arntz, Klokman, & Sieswerda, 2005; Deckers et al., 2015; Herpertz, Gretzer, Mühlbauer, Steinmeyer, & Saß, 1998; Lobbestael and Arntz, 2015; Rosenthal et al., 2016). Other studies, especially when using stimuli not particularly relevant to BPD, report evidence for heightened NA overall but fail to find evidence for NA hyperreactivity in BPD (e.g., Herpertz et al., 2000; Herpertz, Kunert, Schwenger, & Sass, 1999; Kuo & Linehan, 2009). Studies using fMRI report stronger activation of biological correlates of emotion, such as amygdala and insular cortex, in response to pictures of emotional faces or negatively arousing images in individuals with BPD compared to healthy controls (Donegan et al., 2003; Hazlett et al., 2012; Herpertz et al., 2001; Krause-Utz et al., 2012; Prehn et al., 2013). In sum, evidence for NA hyperreactivity in laboratory studies is mixed.

Notably, little is known about positive affect (PA) reactivity and about reactivity to pleasant stimuli. To our knowledge, only a handful

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[1] Abbreviations: NA = Negative Affect; PA = Positive Affect; HAP = High Arousal Positive Affect; LAP = Low Arousal Positive Affect; BPD = Borderline Personality Disorder.

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of studies have experimentally investigated the impact of exposure to pleasant stimuli on PA in BPD. In response to short stories inducing an angry, joyful or neutral mood BPD patients, healthy controls and patients with major depressive disorder showed the same patterns of emotional reactivity (Jacob et al., 2009). In response to pleasant sounds, patients with BPD showed hyporeactivity to pleasant sounds, compared to healthy controls, evidenced both in decreased valence ratings and decreased zygomaticus major reactivity (the facial muscle which is predominant during smiling; Pfaltz et al., 2015). In response to viewing pleasant film clips, patients with BPD showed no significant differences in reactivity of self-reported positive affect when compared to patients with major depression and with healthy controls. Both clinical groups, however, showed a stronger reduction in negative emotions in response to the pleasant film clips (Staebler, Gebhard, Barnett, & Renneberg, 2009). In response to experiencing a rewarding situation (winning a computer game) BPD patients showed a stronger reduction of fear (but similar changes in happiness) compared to non-BPD control groups (Sieswerda, Arntz, & Wolfis, 2005). Taken together, the empirical evidence on PA reactivity from laboratory studies is scarce and mixed, with studies either failing to find altered reactivity or reporting reduced reactivity to pleasant stimuli.

Linehan's biosocial theory (1993) has also been studied in the field of affect dynamics. The DynAffect Model (Kuppens, Oravecz, & Tuerlinckx, 2010) is an overarching model of affect dynamics and has been mapped onto the elements of Linehan's biosocial theory. For BPD, the DynAffect model postulates a negative affective home base (i.e., high negativity) with high levels of affective variability (i.e., instability; Ebner-Priemer et al., 2015). Momentary assessment studies testing the DynAffect model in daily life report higher intensity and instability of affect for individuals with BPD compared to healthy controls (e.g., Ebner-Priemer et al., 2007; Santangelo et al., 2014). Instability in these studies was not contingent on the occurrence of a pleasant or unpleasant event but measured the fluctuation in participants' affect in response to frequent prompts. Individuals with BPD displayed sudden large drops especially from positively valenced mood states (Ebner-Priemer et al., 2007; Santangelo et al., 2014). Results suggest that the instability may be transdiagnostic rather than specific to BPD, given that clinical control groups (i.e., patients with post-traumatic stress disorder and anorexia nervosa) displayed similarly heightened instability (Rosenthal, Fang, & Chapman, 2015; Santangelo et al., 2014). Note that the above-mentioned studies combined these ratings to a single index ranging from negative to positive valence and can thus not differentiate between positive and negative affect. An exception to this approach is a recent momentary assessment study which examined reactivity of both PA and NA in response to daily life events (Houben, Claes, Sleuwaegen, Berens, & Vansteelandt, 2018). That study found that BPD patients (compared to healthy controls) exhibited higher average NA and lower average PA, as well as heightened reactivity of NA in response to disappointment in others, and blunted reactivity of PA in response to positive daily-life events appraised as important.

The study of PA reactivity in the context of BPD deserves more attention for the following reasons. First, increasing our knowledge about whether affect hyperreactivity in BPD is limited to NA, or also applies to PA, will increase our theoretical insight into a core BPD feature. Second, an abundance of studies inspired by Fredrickson's Broaden-and-Build Theory (2001) by now link PA to increased resilience and to higher psychological wellbeing especially after exposure to adversity (e.g., Geschwind et al., 2010; Gloria & Steinhardt, 2016; Rutten et al., 2013; Tugade & Fredrickson, 2004). Other studies suggest that PA promotes a swift recovery from stress (also known as Undoing Effect; Fredrickson, Mancuso, Branigan, & Tugade, 2000; Garland et al., 2010). Both features are especially relevant for people with BPD given their elevated reports of childhood trauma, and the frequent experience of stress and negative emotions (e.g., in response to interpersonal difficulties).

Following the circumplex model of affect (Posner, Russell, & Peterson, 2008; Russell, 1980), research on PA in BPD would benefit from differentiating between high arousal PA (HAP) and low arousal PA (LAP), both in terms of knowledge gain as well as regarding implications for therapy. Current research usually averages various HAP and LAP. The frequently used Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988), for example, measures PA with items on various emotions (both high and low in arousal) combined with items on attention (Schaefer, Nils, Sanchez, & Philippot, 2010). Gaining knowledge about differential reactivity of HAP versus LAP may point to currently underused treatment possibilities. For example, therapy could be augmented with treatment components specifically targeting LAP (e.g. through relaxation training) or HAP (e.g. through imagery focusing on desirable outcomes, etc.). Taxon studies indicate that the underlying structure of BPD likely is dimensional (e.g., Arntz et al., 2009). Given that the current study is the first to investigate differential reactivity of HAP versus LAP in BPD, the evidence for a dimensional structure implies that investigating the influence of *BPD traits* (rather than a BPD diagnosis) is a useful starting point for research on affect reactivity.

The purpose of the current study is to shed light on the relationship between BPD traits and HAP and LAP reactivity in response to a personalized PA-induction. We focused on PA in particular, given the lack of research and the potential to improve therapy by gaining more fine-grained knowledge in this area (Vazquez, 2017). In order to also enable investigation of larger drops in PA, the PA-induction was followed by a stressor. Furthermore, we wanted to test alternative explanations for altered affect reactivity, given evidence for transdiagnostic (rather than BPD-specific) processes (Rosenthal et al., 2015; Santangelo et al., 2014; Staebler et al., 2009). Depressive symptoms were tested because of the high overlap between depression and BPD, and evidence for altered affect reactivity in depression (e.g., Bylsma, Morris, & Rottenberg, 2008; Wichers et al., 2010); mental health care patient (vs. non-patient) and age were tested because they were potentially confounding variables in our study.

Following Linehan's biosocial theory (Crowell et al., 2009; Linehan, 1993) of affective hyperreactivity, the following hypotheses should hold: higher BPD traits will be associated (1) with stronger increases in HAP and LAP, and stronger decreases in NA after PA-induction, and (2) with stronger decreases in HAP and LAP, and stronger increases in NA after the stressor, resulting in an interaction of BPD traits with time. Additionally, given recent research findings on transdiagnostic patterns of affect reactivity, we expect that (3) affective hyperreactivity, if found, will not be specific to BPD traits and thus may be better explainable by patient status or depressive symptoms.

2. Methods

2.1. Participants

Inclusion criteria were: female (because BPD predominantly affects females and to avoid possible gender effects; American Psychiatric Association, 2013), Dutch-speaking, and aged between 18 and 60 years old. To ensure an adequate range of BPD traits, the sample reflected a combination of (a) 73 participants recruited from the general population through advertisements and (b) 35 patients seeking treatment for personality-related problems (recruited from an outpatient mental health care center specialized in personality disorders). Patients were approached for participation when the Structured Clinical Interview for DSM-IV Axis-II Personality Disorders (SCID-II; First, Gibbon, & Spitzer, 1997) indicated presence of three or more BPD symptom criteria during the clinical intake procedures. Patients thus did not necessarily fulfill criteria for BPD. Due to privacy issues, information on the exact clinical diagnoses is not available. The outpatient center staff merely informed us when someone was eligible for participation due to scoring at least 3 BPD symptoms on the SCID-II. Participants received a 15 Euro voucher euro for participation.

Table 1
Participant characteristics and affect at baseline.

	Overall	Non-patients	Patients	p-value (for t-test or Chi2)
	(N = 108)	(N = 73)	(N = 35)	
	M (SD) or n (%)	M (SD) or n (%)	M (SD) or n (%)	
Age	25.35 (8.42)	21.51 (2.73)	33.37 (10.47)	< .001
BPD traits	3.86 (3.26)	2.12 (2.05)	7.49 (2.11)	< .001
Depressive symptoms	15.73 (12.98)	9.30 (8.04)	29.14 (10.91)	< .001
Education highest completed				< .001
Low	1 (0.9%)	0 (0.0%)	1 (2.9%)	
Medium	22 (20.4%)	1 (1.4%)	21 (60%)	
High	14 (13.0%)	8 (10.9%)	6 (17.1%)	
Currently studying	71 (65.7%)	64 (87.7%)	7 (20.0%)	
Work situation				< .001
Employed	15 (13.9%)	9 (23.3%)	6 (17.1%)	
Unemployed	6 (5.6%)	0 (0.0%)	6 (17.1%)	
Student/scholar	71 (65.7%)	64 (87.7%)	7 (20%)	
Social security	16 (14.8%)	0 (0.0%)	16 (45.7%)	
Marital status				.001
Married living together	28 (25.9%)	12 (16.4%)	16 (45.7%)	
Not married/living together	80 (74.1%)	61 (83.6%)	19 (54.3%)	
Psychotropic Medication				< .001
None	69 (63.9%)	58 (79.5%)	11 (31.4%)	
Antidepressants	21 (19.4%)	1 (1.3%)	20 (57.1%)	
Other	18 (16.7%)	14 (19.2%)	4 (11.4%)	
HAP (baseline)	23.47 (7.10)	26.22 (4.94)	17.74 7.55)	< .001
LAP (baseline)	9.81 (3.09)	10.99 (2.46)	7.37 (2.87)	< .001
NA (baseline)	16.02 (7.58)	13.12 (4.53)	22.06 (9.02)	< .001

Note: BPD traits = Borderline Personality Traits. HAP = High Arousal Positive Affect. LAP = Low Arousal Positive Affect. NA = Negative Affect. Other psychotropic medication refers to sleeping pills or benzodiazepines.

Table 1 shows participant characteristics and affect at baseline. Participants were 108 females with a mean age of 25.35 years (range = 18–56). Thirty-two percent of the sample were patients, 68% were non-patients. Nationality was predominantly Dutch (92%), with no significant differences between patient and non-patients. Work status, education level, medication, and living situation were almost collinear with patient status. Also, patients were significantly older than non-patients. Given the significant age differences between patients and non-patients, age was tested as an additional alternative explanation for differential reactivity of affect, next to patient status and depressive symptoms.

2.2. Measures and materials

2.2.1. BPD traits

The McLean Screening Instrument for Borderline Personality Disorder (MSI-BPD) is a brief, 10 item, self-report questionnaire used to measure BPD symptoms (Zanarini et al., 2003; scale range 0–10). For the Dutch version, adequate internal consistency, high specificity and sensitivity, and a high test-retest correlation after four months were found (André, Verschuere, & Lobbestael, 2015; Verschuere & Tibboel, 2011). In the current sample, internal consistency was good (α = .87).

2.2.2. Depressive symptoms

The Centre for Epidemiological Studies-Depression Scale (CES-D, Radloff, 1977; scale range 0–48), was used to assess depressive symptoms during the past week. The Dutch translation has a good internal consistency (Bouma, Ranchor, Sanderman, & van Sonderen, 1995; Schroevers, Sanderman, Van Sonderen, & Ranchor, 2000). In the current sample, internal consistency was excellent (α = .95).

2.2.3. Affect

HAP and LAP were measured using the joviality and serenity scales from the Positive and Negative Affect Schedule-extended (PANAS-X; Watson & Clark, 1999). HAP contained eight items (e.g., happy, joyful, delighted; scale range 8–40; α = .94) and LAP contained three items (calm, relaxed, at ease; scale range 3–15; α = .85). The 10-item NA subscale (e.g., afraid, distressed, hostile; scale range 10–50 [after LN-transformation 2.3–3.7]; α = .91) of the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) was used to measure NA. Item scores were scored on a Likert scale ranging from 1 (very slightly or not at all) to 5 (extremely) and added up per scale. Experimental evidence suggests that the standard PANAS PA scale is less sensitive to mood inductions, possibly because it confounds positive mood with items on attention (Schaefer et al., 2010). Therefore, PA results are provided only in the supplementary analyses, for comparability with other research.

2.2.4. Positive affect induction

Participants were asked to picture themselves in their future life after they had worked hard and all their plans and dreams had come true, and to stay with this image for 1 min. Subsequently, participants wrote about their Best Possible Self for 15 min, followed by 5 min of visualizing this best possible life with as many details as possible. Several studies have shown that this exercise ('Best Possible Self'; King, 2001) reliably increases optimism, and PA (King, 2001; Meevissen, Peters, & Alberts, 2011).

2.2.5. Stressor

Participation in a modified version of the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) was used as a stressor. After a short preparation period, participants were required to deliver a speech (5 min duration) to convince a panel that they were the perfect candidate for their dream job. The speech was followed by a mental arithmetic task (5 min). After a mistake, the experimenter interfered and the participants had to restart. Throughout the procedure, the experimenter acted in a reserved manner. The only difference with the original TSST was that our modified version did not employ a physically present evaluation panel. Instead, we told participants that their speech was being recorded and sent for evaluation by experts working at a recruitment agency. Participants would receive feedback by the end of the week.

The most effective ingredients of successful stress induction, social-evaluative threat and uncontrollability (Dickerson & Kemeny, 2004), were present also in our task. A visual analogue scale (VAS) item ("I feel stressed") with anchors 0 (not at all) to 100 (extremely) was used to assess participants' subjective experience of stress at baseline and right after the stressor.

2.2.6. Filler task

With the goal of capturing participants' attention without intellectual or emotional impact, non-demanding filler tasks (counting color changes on a screen) were used during the two 3-min waiting periods between repeated measures of affect, analogue to the procedure employed by Jacob et al. (2009).

2.3. Procedure

The experiment was approved by the local Ethical review committee of Maastricht University (identification number ECP-137 24_02_2014) and conformed to ethical standards. After providing informed consent, participants were tested individually (duration approximately 1.5 h).

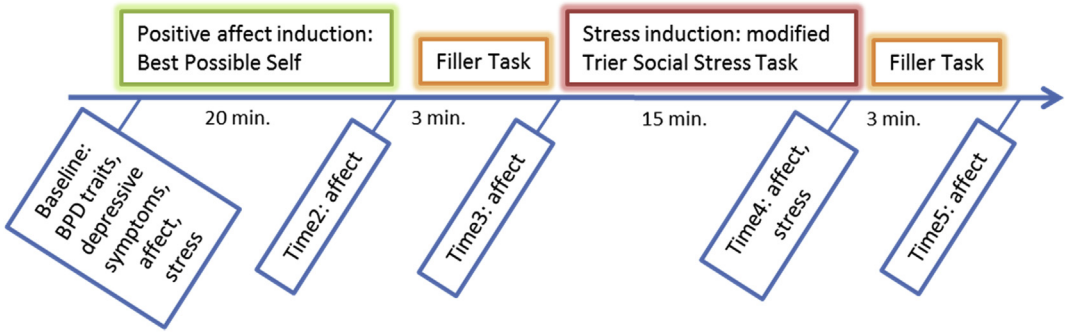

Fig. 1. Experimental procedure: At baseline, BPD traits, depressive symptoms, affect and perceived stress were assessed. Subsequently, participants completed the PA induction exercise and rated their affect (time2). They then engaged in the first filler task for 3 min and rated their affect again (time3). Participants then completed the stress induction task, after which they rated affect and perceived stress (time4), followed by the second 3-min filler task and the last measure of affect (time5).

The timeline of the experiment is depicted in Fig. 1. After the experiment, participants were debriefed and told that no actual film recordings of their speech had been made.

2.4. Power

Standard power calculations are inappropriate for a mixed regression of repeated measures, given that power calculations for mixed models are very complex and depend on many parameters that are unknown in the design stage (Brysbaert & Stevens, 2018). Nevertheless, the power for each effect can to some extent be inferred from the 95% confidence intervals for each parameter (provided in the tables). A narrow (cf. wide) interval means high (cf. low) precision and power.

2.5. Data analysis

Mixed regression for repeated measures with an unstructured covariance pattern was used. Valid reduction to a more parsimonious covariance structure was not possible. The analyses were run in two steps: (1) the initial model included as predictors only *BPD traits* and *time* (using dummy indicator coding with baseline as reference point) and their interaction with each other. (2) To investigate the specificity of the relationship between BPD traits and affect reactivity, the initial model was expanded with the potential confounders *age, patient status* (non-patient vs. patient), *depressive symptoms* and the interaction of these variables with time. Age was included given the significantly higher age in the patient group.

Reducing the number of interaction terms at step 2 was desirable given the high intercorrelations between predictors, see Table 2. In order to identify the best-fitting and most parsimonious model at each step, interactions were dropped one by one (treating the terms of any given predictor with the four time dummies as a single interaction). Main effects were retained in the models. Best-fitting models were identified using Likelihood Ratio (LR) tests based on maximum likelihood (ML) estimation. See Table S1 in the Supplementary Materials for an overview of model comparisons. To correct for multiple testing, we used $\alpha = .01$ instead of 0.05 for final model selection. The reported effects and standard errors are based on restricted maximum likelihood (REML) models (Verbeke & Molenberghs, 2000).

Table 2
Correlations between predictors.

	Patient status	Age	BPD traits
Patient status	–		
Age	.66***	–	
BPD traits	.77***	.51***	–
Depressive symptoms	.72***	.47***	.76***

Note: BPD traits = Borderline Personality Traits.***$p < .001$.

Dependent variables were HAP, LAP, and NA (with PA reported in the supplementary material). Due to non-normality of residuals of NA, we analyzed LN-transformed NA. The results were the same as for non-transformed NA with respect to direction and presence of predictor effects.

3. Results

3.1. Main effects

Unadjusted for patient status, age, and depression, higher BPD traits were associated with significantly lower HAP and LAP, and significantly higher NA across time points (see Table 3, Step 1).

Adjusted for patient status, age, and depressive symptoms depressive symptoms were strongly associated with lower HAP and LAP, and higher NA across time points (see Table 3, Step 2), whereas the effects of BPD traits and patient status were not significant.

Regarding the main effects of PA-induction and stressor *across participants*, the results of step1 and step2 models correspond with each other. Table 3 shows significant yet short-lived increases in HAP right after PA-induction, with a return to baseline after 3 min. PA-induction did not lead to significant increases in LAP. NA was significantly lower 3 min after but not directly after PA-induction. Relative to baseline, the stressor was associated with significant decreases in HAP and LAP across participants, both directly after (time4) as well as 3 min later (time5), and with short-lived NA increases across participants (significant at time4 but not time5).[2]

3.2. Step 1: model unadjusted for age, patient status and depression

Table 4 shows results of BPD trait by time models for HAP, LAP, and NA. Fig. 2 shows plots of predicted values for all outcomes, with separate lines for subgroups with high, medium, or low BPD traits (based on a tertile split). The tertile split was used only for visualization purposes; all analyses used continuous variables. Fig. S1 in the Supplementary Materials shows corresponding plots of observed values, Fig. S4 shows individual variation using spaghetti-plots of observed values.

After PA-induction, higher BPD traits were associated with higher increases in LAP, and with higher decreases in NA (indicating hyperreactivity to PA-induction). Effects were immediately present for LAP and delayed for NA. Higher BPD traits may also be associated with a weaker HAP response to PA-induction, though the interaction of BPD

[2] A manipulation check indicated that the subjective experience of stress increased significantly across participants (from M = 30.88, SD = 26.77 at baseline to M = 51.48, SD = 28.62 after the stressor; $t(94) = 6.27$; $p < .001$). Note that the manipulation check for the BPS is increase in PA. Given that the Best Possible Self task followed baseline, the manipulation check for the Best Possible Self is thus part of the outcome data.

Table 3
Fixed effects estimates for main effects models at step 1 and step 2.

Parameter	HAP		LAP		NA (LN-transformed)	
	B	95% CI	*B*	95% CI	*B*	95% CI
Step 1						
Intercept	28.37***	[26.78, 29.96]	11.56***	[10.91, 12.21]	2.47***	[2.39, 2.55]
BPD traits	−1.27***	[-1.57, −.97]	-.45***	[-.57, −.34]	.06***	[.04, .07]
Time2	2.77***	[1.83, 3.71]	.13	[-.33, .59]	-.03	[-.08, .02]
Time3	.25	[-.82, −2.26]	.29	[-.10, .69]	-.10***	[-.15, −.05]
Time4	−3.65***	[-5.04, −2.26]	−2.84***	[-3.43, −2.25]	.29***	[.20, .38]
Time 5	−2.39***	[-3.70, −1.08]	−1.03***	[-1.58, −.48]	.04	[-.04, .12]
Step 2						
Intercept	28.42***	[25.27, 31.56]	11.87***	[10.62, 13.12]	2.46***	[2.31, 2.61]
BPD traits	-.25	[-.70, .19]	-.09	[-.26, .09]	.00	[-.03, .02]
Patient status	-.59	[-3.83, 2.65]	.14	[-1.13, 1.41]	.12	[-.04, .28]
Age	.07	[-.06, .20]	.02	[-.03, .07]	.00	[-.01, .00]
Dep	-.35***	[-.45, −.25]	-.14***	[-.18, −.10]	.02***	[.01, .02]
Time2	2.76***	[1.82, 3.70]	.12	[-.34, .58]	-.03	[-.08, .02]
Time3	.22	[-.84, 1.29]	.29	[-.10, .68]	-.10***	[-.15, −.05]
Time4	−3.69***	[-5.07, −2.30]	−2.88***	[-3.47, −2.29]	.29***	[.21, .38]
Time5	−2.42***	[-3.73, −1.11]	−1.06***	[-1.61, −.50]	.04	[-.03, .12]

Note: Patient status was coded as 0 (non-patients) and 1 (patients). HAP = High Arousal Positive Affect. LAP = Low Arousal Positive Affect. NA = Negative Affect. BPD traits = Borderline Personality traits. Dep = depressive symptoms. Time2 = right after positive affect induction. Time3 = 3 min after positive affect induction. Time4 = right after stressor. Time5 = 3 min after stressor. *SE* per *B* is approx. ¼-th the confidence interval width. Scale ranges: BPD traits 0–10, Dep 0–48, HAP 8–40, LAP 3–15, NA (LN-transformed) 2.3–3.7.
$^{*}p \leq .05$. $^{**}p < .01$. $^{***}p < .001$.

Table 4
Fixed effects estimates for initial models, unadjusted for patient status, age, and depression (step 1).

Parameter	HAP		LAP		NA (LN-transformed)	
	B	95% CI	*B*	95% CI	*B*	95% CI
Intercept	28.38***	[26.67, 3.10]	12.01***	[11.27, 12.75]	2.42***	[2.32, 2.51]
BPD traits	−1.27***	[-1.61, −.93]	-.57***	[-.71, −.42]	.07***	[.05, .09]
time2	3.98***	[2.55, 5.40]	-.60	[-1.30, .09]	.02	[-.06, .10]
time3	.45	[-1.20, 2.11]	-.33	[-.92, .25]	-.01	[-.09, .07]
time4	−3.27***	[-5.37, −1.16]	−3.39***	[-4.27, −2.50]	.36***	[.23, .49]
time5	−2.45*	[-4.45, −.46]	−1.64***	[-2.47, −.81]	.11	[-.01, .22]
BPD traits x time2	-.32*	[-.60, −.03]	.19**	[.05, .33]	-.01	[-.03, .00]
BPD traits x time3	-.05	[-.38, .28]	.16**	[.05, .28]	-.02**	[-.04, −.01]
BPD traits x time4	-.11	[-.55, .33]	.14	[-.04, .33]	-.02	[-.05, .01]
BPD traits x time5	.02	[-.39, .44]	.16	[-.01, .33]	-.02	[-.04, .01]

Note: CI = Confidence Interval. HAP = High Arousal Positive Affect. LAP = Low Arousal Positive Affect. NA = Negative Affect. BPD traits = Borderline Personality Traits. Time2 = right after positive affect induction. Time3 = 3 min after positive affect induction. Time4 = right after stressor. Time5 = 3 min after stressor. *SE* per *B* is approx. ¼-th the confidence interval width. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

traits with time2 failed to reach our more stringent significance level of $p = 0.01$, and the interaction of BPD traits with time3 was not significant.

There was no evidence for BPD trait-dependent hyperreactivity of HAP, LAP, or NA in response to the stressor. Fig. 2 shows how changes in HAP, LAP, and NA from time3 to time4 and from time4 to time5 were similar for participants with high, medium, and low BPD traits.[3]

3.3. Step 2: adjusting for age, patient status and depressive symptoms

In the second step, the models for each outcome were extended with the variables age, patient status, depressive symptoms, and the interaction of these variables with time. Non-significant interaction terms were removed step by step because of the high correlations between independent variables. Likelihood ratio (Chi2) tests of the reduced versus preceding model were used to select the best-fitting model. Table S1 in the Supplementary Materials shows the goodness of fit for all possible models per outcome. Table 5 shows fixed effects estimates of the best-fitting model per outcome (not showing the model for untransformed NA because the skewness of this variable leads to invalid confidence intervals and p-values). Fig. 3 shows plots of predicted values for HAP, LAP, and NA (LN-transformed and untransformed), with the predicted values as computed from the models in Table 5.[4] Fig. S2 in the Supplementary Materials shows corresponding plots of observed values, Fig. S5 shows individual variation using spaghetti-plots of observed values.

For *HAP*, the model with patient status by time interaction as the only interaction was the best-fitting model. Compared to non-patients, patients showed a blunted HAP response right after PA-induction (see the missing peak at time2 for patients in Fig. 3a; marginally significant, $p = .012$). There were no significant differences at other time points.

For *LAP and NA*, the model with only the interaction between depressive symptoms and time fitted best. This means that the interaction

[3] Thirteen participants (12 patients and 1 non-patient) refused the stress test and dropped out after time 3. Their available data were included into the analyses with mixed regression, thus preventing bias arising from selective drop-out (Verbeke & Molenberghs, 2000).

[4] Figs. S1 and S2 in the Supplementary materials show plots of the observed values for these outcomes. Observed and predicted plots were highly similar, indicating a good model fit.

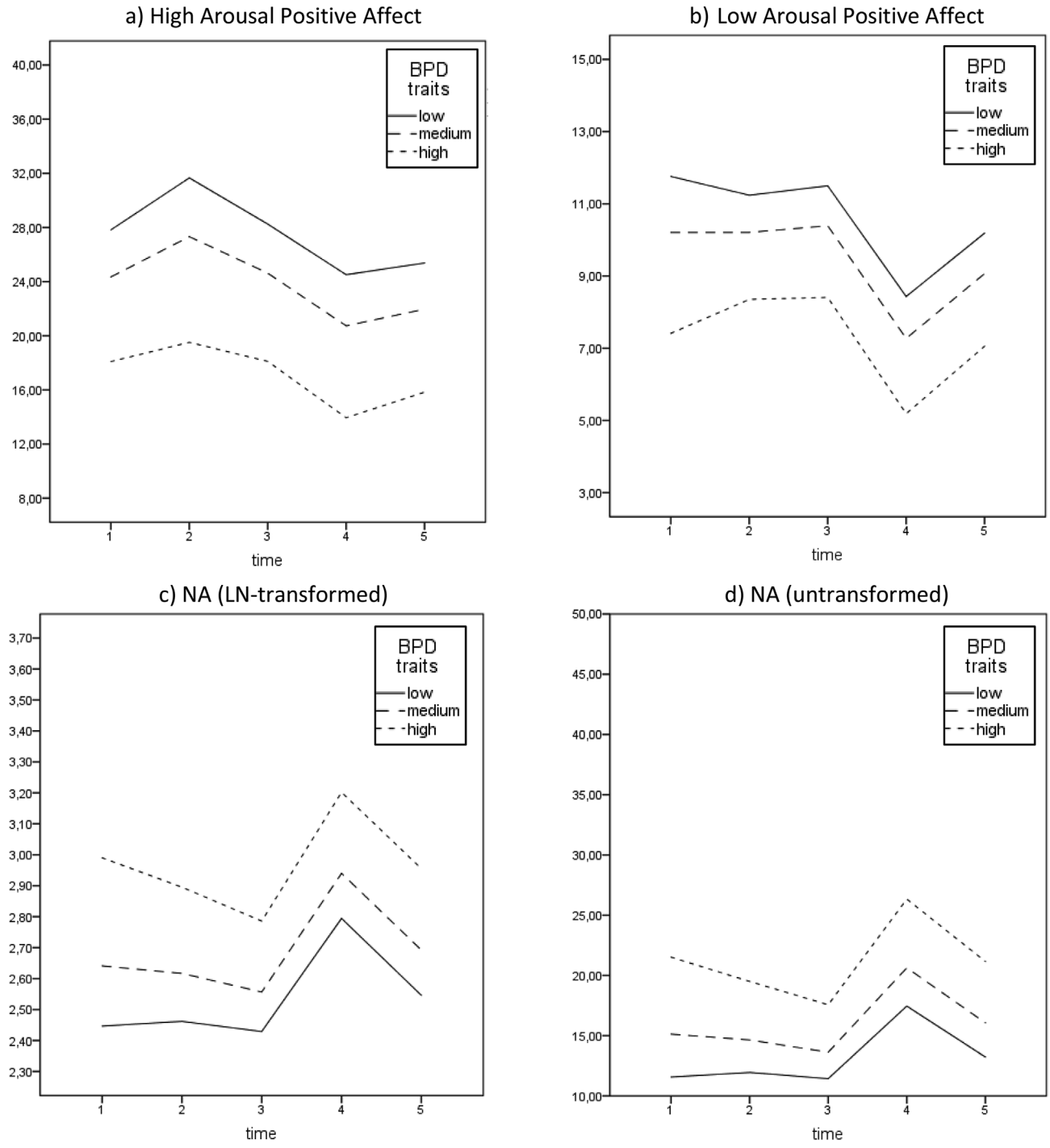

Fig. 2. Mean predicted values of High Arousal Positive Affect (HAP), Low Arousal Positive Affect (LAP) and Negative Affect (NA, LN-transformed and untransformed) per time point. Predicted values are based on the best-fitting models at step 1, thus including the predictors BPD traits and time, as well as their interaction with each other. Lines for high, medium, and low BPD traits are based on a tertile split (mean BPD traits per tertile were 8.09, 3.18, and 0.44, respectively). Time1 = baseline, time2 = after positive affect induction, time3 = 3 min after positive affect induction, time4 = after stress induction, time5 = 3 min after stress induction. Scale ranges: BPD traits 0–10, HAP 8–40, LAP 3–15, NA (LN-transformed) 2.30–3.69, NA 10–50.

of depression by time predicted reactivity of LAP and NA significantly better than models with interactions of either BPD traits by time or patient status by time, and that interactions of either BPD traits or patient status with time were non-significant when included alongside depressive symptoms by time interactions. The pattern of findings from the models with BPD traits by time, or patient status by time interaction, were similar to the pattern found for the best model with depression by time interaction, indicating a quantitative rather than a qualitative difference.

After PA-induction, higher depressive symptoms predicted higher reactivity of LAP and NA, see Table 5. Fig. 3b, c and d show that only the subgroup of participants with the highest depressive symptoms (based on a tertile split) experienced an increase in LAP and a decrease in NA in response to PA-induction, while the low and medium depressive symptoms groups experienced no change in LAP.

Following the stressor, participants with high depressive symptoms showed a weaker decrease in LAP and a weaker increase in NA, relative to baseline, compared to participants with low or medium depressive

Table 5
Fixed effects estimates for best-fitting models after inclusion of age, patient status, and depressive symptoms (step 2).

	HAP		LAP		NA (LN-transformed)	
Parameter	*B*	95% CI	*B*	95% CI	*B*	95% CI
Intercept	28.56***	[25.40, 31.73]	12.37***	[11.09, 13.66]	2.41***	[2.25, 2.56]
BPD traits	-.25	[-.70, .19]	-.09	[-.26, .09]	.00	[-.03, .02]
Patient status	-.96	[-4.37, 2.45]	.16	[-1.12, 1.43]	.12	[-.04, .27]
Age	.07	[-.06, .20]	.02	[-.03, .07]	.00	[-.01, .00]
Depressive symptoms	-.35***	[-.45, −.25]	-.17***	[-.22, −.13]	.02***	[.02, .03]
Time2	3.55***	[2.44, 4.66]	-.65	[-1.35, .06]	.03	[-.04, .11]
Time3	.08	[-1.21, 1.37]	-.65	[-1.22, −.08]	.04	[-.04, .11]
Time4	−3.80***	[-5.43, −2.17]	−3.69***	[-4.58, −2.80]	.44***	[.31, .56]
Time5	−2.69***	[-4.23, −1.15]	−1.77***	[-2.62, −.92]	.17**	[.05, .28]
Patient status x time2	−2.55*	[-4.53, −.56]				
Patient status x time3	.44	[-1.87, 2.74]				
Patient status x time4	.32	[-2.86, 3.49]				
Patient status x time5	1.02	[-1.92, 3.95]				
Dep x time2		.05**	[.01, .08]	-.004*		[-.01, .00]
Dep x time3		.06***	[.03, .09]	-.01***		[-.01, .00]
Dep x time4		.05*	[.01, .10]	-.01**		[-.02, .00]
Dep x time5		.04*	[.00, .09]	-.01**		[-.01, .00]

Note: CI = Confidence Interval. HAP = High Arousal Positive Affect. LAP = Low Arousal Positive Affect. NA = Negative Affect. BPD traits = Borderline Personality Traits. Patient status was coded as 0 (non-patients) and 1 (patients). Time2 = right after positive affect induction. Time3 = 3 min after positive affect induction. Time4 = right after stress induction. Time5 = 3 min after stress induction. *SE* per *B* is approx. ¼-th the confidence interval width. *$p \leq .05$. **$p < .01$. ***$p < .001$.

symptoms (see Table 5; marginally significant for LAP, $p < .05$ but > 0.01). Fig. 3b, c and d show parallel lines between time3 and time4, therefore weaker reactivity after stress is attributable to higher gains after PA-induction.

4. Discussion

Leading models on BPD designate a central role to affective hyperreactivity (American Psychiatric Association, 2013; Crowell et al., 2009; Kuppens et al., 2010; Linehan, 1993). Largely, these theories are silent about whether this would apply to positive as well as negative affect, although Linehan does specifically presume the presence of such an overall affective dysregulation in BPD. The current study, to the best of our knowledge, is the first to test the relationship between BPD traits and the evolution of high and low arousal PA (HAP and LAP) as well as NA throughout a PA-induction followed by a stressor. We also investigated the robustness of associations between BPD traits and affect reactivity by testing alternative explanations (i.e., patient vs. non-patient status, age, and depressive symptoms) and found that – in line with recent transdiagnostic research findings – altered affect reactivity was not specific to BPD traits.

4.1. At first glance: BPD traits and affect reactivity

In response to PA-induction, higher BPD traits were associated with a dampened HAP increase (indicative of reduced affect reactivity; marginally significant) and a significantly stronger LAP increase and NA decrease (indicative of hyperreactivity to positive stimuli). We did not find hyperreactivity of HAP, LAP, or NA in response to the stressor, relative to baseline.

4.2. At second glance: No evidence for specificity

When age, patient status, depressive symptoms, and their interactions with time were added as independent variables and these complex models were then reduced to prevent collinearity, we found no evidence for altered affect reactivity as a specific feature of BPD. Reactivity of HAP was best predicted by an interaction between patient status and time, while reactivity of LAP and NA was best predicted by the interaction between depressive symptoms and time.

Patients experienced a blunted HAP increase after PA-induction,[5] indicative of *hypo*reactivity. The increase of HAP in patients was only 28% of the increase in non-patients. Reactivity of HAP from baseline to after the stressor (which was preceded by the PA-induction), was not different for patients and non-patients.

Higher depressive symptoms were associated with (1) stronger improvement in LAP and NA after PA-induction, and (2) blunted deterioration of LAP[4] and NA after the stressor, relative to baseline. It could be argued that participants with higher depressive symptoms had more room to improve and less room to deteriorate on LAP and NA, given that their baseline LAP was lower and their baseline NA was higher than that of participants with lower depressive symptoms. On the other hand, this baseline difference is unlikely to fully account for our findings, given that reactivity of HAP to the PA-induction was blunted even though HAP was also considerably lower in participants with higher depressive symptoms. Another possibility is that depressive symptoms (or maybe medication used to treat these symptoms) caused blunted LAP and NA reactivity to the stressor as well as blunted HAP reactivity to the PA-induction. The phenomenon of blunted reactivity to both positively and negatively valenced stimuli in people with major depressive disorder has been termed emotion context insensitivity (Bylsma et al., 2008). Yet again, the fact that participants with high depressive symptoms displayed *stronger* reactivity for LAP and NA after PA-induction discounts emotion context insensitivity as an overall coherent explanation for our findings. In our sample, where the stressor followed PA-induction, participants with high depressive symptoms experienced only 30–60% of the mood deterioration experienced by participants with low depressive symptoms (depending on time point and outcome variable). Possibly, the higher gains after the PA-induction exercise may have conferred a protective influence against the mood-deteriorating effects of stress on NA. Further research will be necessary to shed light on whether the PA-induction indeed may have had a beneficial effect on resilience against stress.

Taken together, depressive symptoms were the strongest predictor of hyperreactivity of LAP and NA in response to PA-induction and stressor, while being a mental health care patient best predicted HAP hyporeactivity after PA-induction. These findings either suggests that altered affect reactivity is transdiagnostic in nature (i.e., also related to

[5] Marginally significant: $p > .01$ but < 0.05.

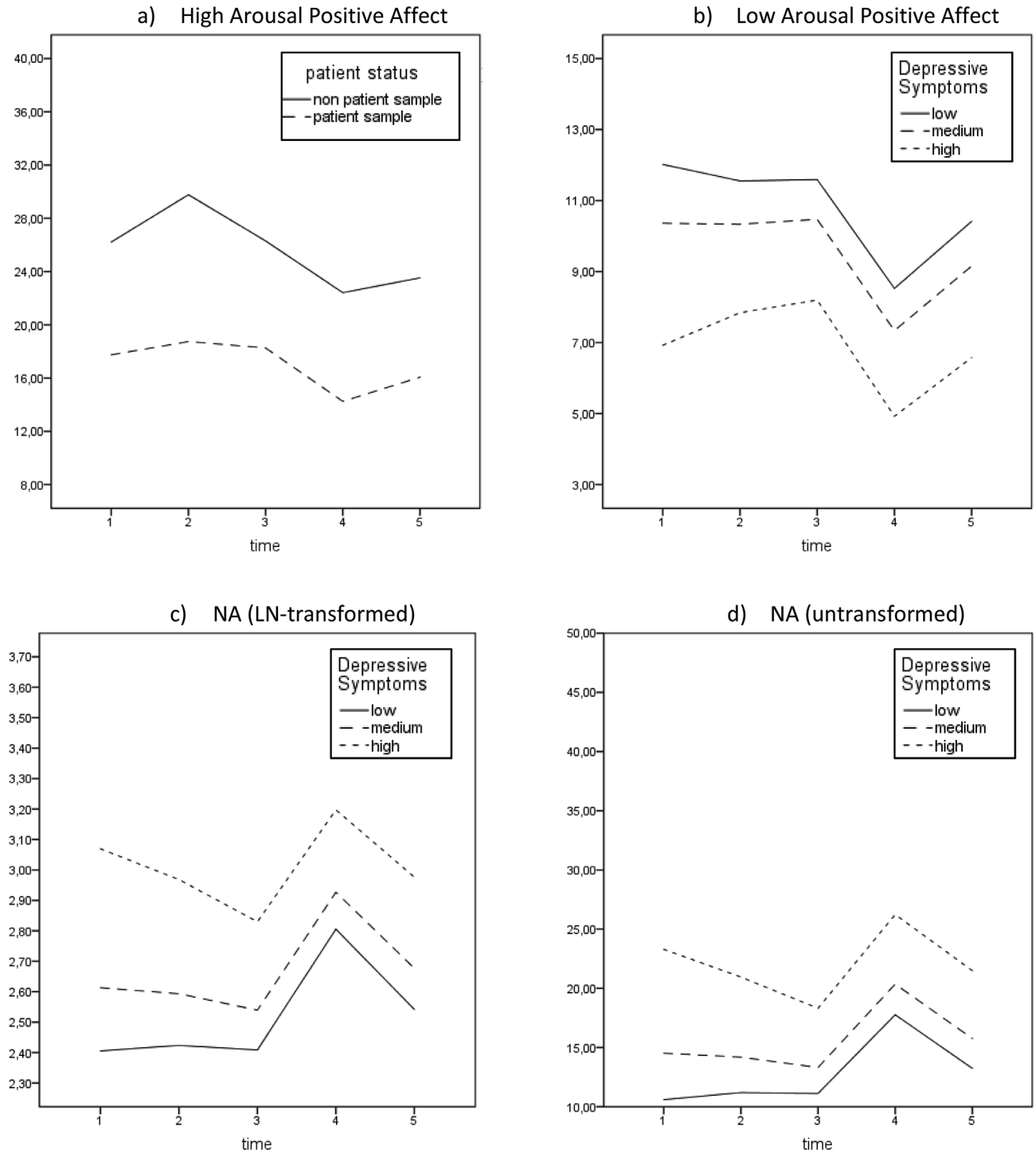

Fig. 3. Mean predicted values of High Arousal Positive Affect, Low Arousal Positive Affect, and Negative Affect (NA; LN-transformed and untransformed) per time point. Predicted values are based on the best-fitting models at step 2 as reported in Table 5, thus including main effects of patient status, BPD traits, age, depression and time, and interaction terms patient status by time (for High Arousal Positive Affect), or depression by time (for Low Arousal Positive Affect and NA). Lines for high, medium, and low depressive symptoms are based on a tertile split (mean depressive symptoms per tertile were 31.7, 12.6, and 3.7, respectively). Time1 = baseline, time2 = after positive affect induction, time3 = 3 min after positive affect induction, time4 = after stressor, time5 = 3 min after stressor. Scale ranges: BPD traits 0–10, HAP 8–40, LAP 3–15, NA (LN-transformed) 2.30–3.69, NA 10–50.

depressive disorders and psychopathology other than BPD), or that depressive symptoms and patient status mediate the effects of BPD traits on affect. Furthermore, the current study highlights the importance of an increased research focus on affect reactivity to pleasant stimuli.

4.3. Clinical implications

Our results imply that people with heightened psychopathology – whether characterized by patient status, borderline traits, or depressive symptoms – may especially benefit from exercises focused on PA (in terms of immediate improvements in LAP and NA). PA may therefore represent a valuable and currently under-used target for psychotherapy

(Craske et al., 2019; Dunn, 2019; Geschwind, Arntz, Bannink, & Peeters, 2019; Sewart et al., 2019). Reflecting on the Best Possible Self (rather than recalling positive memories) may be a particularly effective way of stimulating positive affect for people with heightened psychopathology, given that a recent study found that adopting a more analytic, reflective perspective (rather than merely recounting positive events) led to increased improvements in both PA and NA for individuals with major depression (Pfaltz et al., 2017).

Our findings indicate that BPD traits should certainly not be considered contra-indicative for a focus on PA. Positive cognitive-behavioral therapy (Bannink, 2012) or strengthening positive modes in schema therapy (Kellogg & Young, 2006) may consequently represent a patient-friendly way to increase PA. Gaining more knowledge about how to stimulate both HAP and LAP best in patients with BPD remains an important area for future research. The fact that higher psychopathology was associated with a blunted HAP response to the PA-induction (the Best Possible Self task) may suggest difficulties in picturing a future in which all went well. Another option is that certain topics may induce more HAP for people with high psychopathology than other topics. Research on mood repair suggests that participants who spontaneously chose to recall social memories experienced more mood-repair than participants who spontaneously chose to recall achievement-related memories (Seebauer et al., 2016). That study also found that it was possible to target specific positive emotions by experimentally varying the content of a memory (Seebauer et al., 2016). Similarly, content analysis of Best Possible Self material found that content related to family and friends was more likely to increase optimism than content related to physical health or financial goals (Boselie, 2017). Restricting content to these topics may thus be more successful in elevating HAP.

Alternatively, improving LAP may be the most pressing (and hence possibly most useful) issue for people with high psychopathology. On the other hand, momentary assessment studies found that reward experience (the increase of PA after pleasant activities) contributes to increased resilience against affective symptoms in the face of major stressful life events (Geschwind et al., 2010; Rutten et al., 2013). Importantly, the items in the PA scales of these studies tended to focus on HAP (i.e., happy, enthusiastic, cheerful), rather than on LAP. For example, relaxed, one of the three items measuring LAP, was not included in these studies' PA scales due to low factor loadings; Geschwind et al., 2010; Geschwind, Peeters, Drukker, van Os, & Wichers, 2011). According to Frijda (1986, p. 89), HAP encourages an "unasked-for readiness to engage in whatever interaction presents itself, and in part readiness to engage in enjoyments". If playfulness and exploration underlie good long-term mental health, then training how to explore, play, and enjoy may be a useful psychotherapy strategy.

4.4. Limitations and recommendations for future research

Strengths of the study include the personalized (rather than standardized) PA-induction and stress tasks, as well as the differentiation between high and low-arousal PA. Limitations include the following: First, the sample was a convenience sample amplified with patients from an outpatient Personality Disorder treatment unit, rather than patients with full-blown BPD. Even though use of such a mixed sample is in line with taxon studies evidencing the dimensional underlying structure of BPD (Arntz et al., 2009), replication in a sample with BPD patients would be useful. Second, as described in the 'measures and materials' section, the stress task deviated from the original TSST in that no physically present panel was employed. The manipulation check did, however indicate that the stress induction was successful. Third, patients were more likely to refuse participation in the stress task than non-patients, thus potentially introducing a confounding effect. We did, however, use mixed (multi-level) modeling, thus limiting damage due to selective dropout (Schafer & Graham, 2002; Verbeke & Molenberghs, 2000). Mixed modeling corrects for dropout as much as possible, such that dropout related to our predictors (age, patient status, BPD traits, depressive symptoms) or to outcome levels at time points preceding dropout could not bias our results. Fourth, medication may have influenced affect reactivity, because taking psychotropic medication was correlated with severity of psychopathology. Finally, given that the stressor was a speech task, it may have been useful to additionally measure anxiety as an alternative predictor.

A general recommendation for future research is to continue exploring affect reactivity to pleasant stimuli in BPD through experimental studies, given that so little is known about this topic. Gaining more knowledge about how to stimulate HAP in people vulnerable to mental health problems (be it through heightened BPD traits or depressive symptoms) may point to currently underused treatment paths. Running a content-analysis of self-generated positive material (like the Best Possible Self essays used in the current study) to find out whether content differs depending on participants' mental health, and how content differentially relates to increases in HAP versus LAP could be a first step. If certain themes are more likely to increase HAP, future research may investigate the effects of restricting the content to these themes. Future research may also investigate effects of more BPD-specific positive triggers (i.e., visualizing stable relationships, being accepted and included). In order to allow conclusions about whether or not PA-induction increases resilience to stress, future research should randomize to neutral or PA-induction before exposure to stress or swap the order of PA-induction and stressor.

Overall, we strongly recommend differentiating between high and low arousal PA in future studies given the different trajectories for HAP and LAP found in the current study, in which participants with higher psychopathology responded to the PA-induction especially with an increase in LAP, whereas participants with lower psychopathology responded with an increase in HAP but not LAP. The potentially different impact of high versus low arousal PA on mental well-being needs further examination, especially in longer-term-studies. Finally, the strong (potentially mediating) influence of depressive symptoms on affect reactivity in the current sample points to the importance of transdiagnostic research, rather than research focused on BPD features selectively.

5. Conclusions

Three main conclusions emerge from the current study: (1) Differential affect reactivity was not specific to BPD traits but seemed to be related to psychopathology in general (i.e., co-occurring depressive symptoms and being a mental health care patient). (2) Higher psychopathology was associated with a blunted HAP increase, but a stronger LAP increase in response to PA-induction, suggesting a need for more fine-grained investigation of PA reactivity, at least differentiating between HAP and LAP. (3) Reactivity of LAP and NA to stress was significantly blunted for participants with higher compared to lower psychopathology in our study, in which exposure to stress was preceded by PA-induction. Our results therefore suggest the necessity to further explore whether a focus on PA in psychotherapy might enhance resilience to stress. More research is also needed regarding the optimal stimulation of HAP and LAP, as well as their long-term effects on reactivity to stressors.

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Declaration of interest

The authors declare that they have no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jbtep.2019.101497.

References

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). (Washington, DC).

André, J. A., Verschuere, B., & Lobbestael, J. (2015). Diagnostic value of the Dutch version of the McLean screening instrument for BPD (MSI-BPD). *Journal of Personality Disorders, 29*(1), 71–78. https://doi.org/10.1521/pedi_2014_28_148.

Arntz, A., Bernstein, D., Gielen, D., van Nieuwenhuyzen, M., Penders, K., Haslam, N., et al. (2009). Taxometric evidence for the dimensional structure of cluster-C, paranoid, and borderline personality disorders. *Journal of Personality Disorders, 23*(6), 606–628. https://doi.org/10.1521/pedi.2009.23.6.606.

Arntz, A., Klokman, J., & Sieswerda, S. (2005). An experimental test of the schema mode model of borderline personality disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 36*(3), 226–239. https://doi.org/10.1016/j.jbtep.2005.05.005.

Bannink, F. (2012). *Practicing positive CBT: From reducing distress to building success.* Chichester, UK: John Wiley & Sons, Ltd.

Boselie, J. (2017). *Filling the glass: Optimism as a protective factor against executive functioning impairments*(PhD thesis) Maastricht: University Press Maastricht. Retrieved from https://cris.maastrichtuniversity.nl/portal/files/16087389/c5763.pdf.

Bouma, J., Ranchor, A. V., Sanderman, R., & van Sonderen, E. (1995). *Het meten van symptomen van depressie met de CES-D, een handleiding. [Measuring symptoms with the CES-D, a manual.] NCG Reeks Meetinstrumenten, Vol. 6*.

Brysbaert, M., & Stevens, M. (2018). Power analysis and effect size in mixed effects models: A tutorial. *Journal of Cognition, 1*(1), 1–20. https://doi.org/10.5334/joc.10.

Bylsma, L. M., Morris, B. H., & Rottenberg, J. (2008). A meta-analysis of emotional reactivity in major depressive disorder. *Clinical Psychology Review, 28*(4), 676–691. https://doi.org/10.1016/j.cpr.2007.10.001.

Craske, M. G., Meuret, A. E., Ritz, T., Treanor, M., Dour, H., & Rosenfield, D. (2019). Positive affect treatment for depression and anxiety: A randomized clinical trial for a core feature of anhedonia. *Journal of Consulting and Clinical Psychology, 87*(5), 457–471. https://doi.org/10.1037/ccp0000396.

Crowell, S. E., Beauchaine, T. P., & Linehan, M. M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending linehan's theory. *Psychological Bulletin, 135*(3), 495–510. https://doi.org/10.1037/a0015616.

Deckers, J. W. M., Lobbestael, J., van Wingen, G. A., Kessels, R. P. C., Arntz, A., & Egger, J. I. M. (2015). The influence of stress on social cognition in patients with borderline personality disorder. *Psychoneuroendocrinology, 52*(1), 119–129. https://doi.org/10.1016/j.psyneuen.2014.11.003.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*(3), 355–391. https://doi.org/10.1037/0033-2909.130.3.355.

Donegan, N. H., Sanislow, C. A., Blumberg, H. P., Fulbright, R. K., Lacadie, C., Skudlarski, P., et al. (2003). Amygdala hyperreactivity in borderline personality disorder: Implications for emotional dysregulation. *Biological Psychiatry, 54*(11), 1284–1293. https://doi.org/10.1016/S0006-3223(03)00636-X.

Dunn, B. D., Widnall, E., Reed, N., Taylor, R., Owens, C., Spencer, A., et al. (2019). Evaluating augmented depression therapy (ADepT): Study protocol for a pilot randomised controlled trial. *Pilot and Feasibility Studies, 5*(1), 63. https://doi.org/10.1186/s40814-019-0438-1.

Ebner-Priemer, U., Houben, M., Santangelo, P., Kleindienst, N., Tuerlinckx, F., Oravecz, Z., et al. (2015). Unraveling affective dysregulation in borderline personality disorder: A theoretical model and empirical evidence. *Journal of Abnormal Psychology, 124*(1), 186–198. https://doi.org/10.1037/abn0000021.

Ebner-Priemer, U., Kuo, J., Kleindienst, N., Welch, S. S., Reisch, T., Reinhard, I., et al. (2007). State affective instability in borderline personality disorder assessed by ambulatory monitoring. *Psychological Medicine, 37*(07), 961. https://doi.org/10.1017/S0033291706009706.

First, M., Gibbon, M., & Spitzer, R. (1997). *Structured clinical Interview for DSM-IV Axis II personality disorders (SCID-II).* American Psychiatric Press, Inc.

Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist, 56*(3), 218–226. https://doi.org/10.1037//0003-066X.56.3.218.

Fredrickson, B. L., Mancuso, R. A., Branigan, C., & Tugade, M. M. (2000). The undoing effect of positive emotions. *Motivation and Emotion, 24*(4), 237–258. https://doi.org/10.1023/a:1010796329158.

Frijda, N. (1986). *The emotions.* Cambridge: Cambridge University Press. https://doi.org/10.1093/0199253048.001.0001.

Garland, E. L., Fredrickson, B., Kring, A. M., Johnson, D. P., Meyer, P. S., & Penn, D. L. (2010). Upward spirals of positive emotions counter downward spirals of negativity: Insights from the broaden-and-build theory and affective neuroscience on the treatment of emotion dysfunctions and deficits in psychopathology. Clinical Psychology Review. https://doi.org/10.1016/j.cpr.2010.03.002.

Geschwind, N., Arntz, A., Bannink, F., & Peeters, F. (2019). Positive cognitive behavior therapy in the treatment of depression: A randomized order within-subject comparison with traditional cognitive behavior therapy. *Behaviour Research and Therapy, 116*, 119–130. https://doi.org/10.1016/j.brat.2019.03.005.

Geschwind, N., Peeters, F., Drukker, M., van Os, J., & Wichers, M. (2011). Mindfulness training increases momentary positive emotions and reward experience in adults vulnerable to depression: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 79*(5), 618–628. https://doi.org/10.1037/a0024595.

Geschwind, N., Peeters, F., Jacobs, N., Delespaul, P., Derom, C., Thiery, E., et al. (2010). Meeting risk with resilience: High daily life reward experience preserves mental health. *Acta Psychiatrica Scandinavica, 122*(2), 129–138. https://doi.org/10.1111/j.1600-0447.2009.01525.x.

Gloria, C. T., & Steinhardt, M. A. (2016). Relationships among positive emotions, coping, resilience and mental health. *Stress and Health, 32*(2), 145–156. https://doi.org/10.1002/smi.2589.

Hazlett, E. A., Zhang, J., New, A. S., Zelmanova, Y., Goldstein, K. E., Haznedar, M. M., et al. (2012). Potentiated amygdala response to repeated emotional pictures in borderline personality disorder. *Biological Psychiatry, 72*(6), 448–456. https://doi.org/10.1016/j.biopsych.2012.03.027.

Herpertz, S., Dietrich, T. M., Wenning, B., Krings, T., Erberich, S. G., Willmes, K., et al. (2001). Evidence of abnormal amygdala functioning in borderline personality disorder: A functional MRI study. *Biological Psychiatry, 50*(4), 292–298. https://doi.org/10.1016/S0006-3223(01)01075-7.

Herpertz, S., Gretzer, A., Mühlbauer, V., Steinmeyer, E. M., & Saß, H. (1998). Experimenteller Nachweis mangelnder Affektregulation bei Patientinnen mit selbstschädigendem Verhalten [Experimental evidence of lack of affect regulation in female patients with self-damaging behaviour]. *Nervenarzt, Der, 69*(5), 410–418. https://doi.org/10.1007/s001150050290.

Herpertz, S., Kunert, H. J., Schwenger, U. B., & Sass, H. (1999). Affective responsiveness in borderline personality disorder: A psychophysiological approach. *American Journal of Psychiatry, 156*(10), 1550–1556. https://doi.org/10.1176/ajp.156.10.1550.

Herpertz, S., Schwenger, U. B., Kunert, H. J., Lukas, G., Gretzer, U., Nutzmann, J., et al. (2000). Emotional responses in patients with borderline as compared with avoidant personality disorder. *Journal of Personality Disorders, 14*(4), 339–351. https://doi.org/10.1521/pedi.2000.14.4.339.

Houben, M., Claes, L., Sleuwaegen, E., Berens, A., & Vansteelandt, K. (2018). Emotional reactivity to appraisals in patients with a borderline personality disorder: A daily life study. *Borderline Personality Disorder and Emotion Dysregulation, 5*(1), 18. https://doi.org/10.1186/s40479-018-0095-7.

Jacob, G. a, Hellstern, K., Ower, N., Pillmann, M., Scheel, C. N., Rüsch, N., et al. (2009). Emotional reactions to standardized stimuli in women with borderline personality disorder. *The Journal of Nervous and Mental Disease, 197*(11), 808–815. https://doi.org/10.1097/NMD.0b013e3181bea44d.

Kellogg, S. H., & Young, J. E. (2006). Schema therapy for borderline personality disorder. *Journal of Clinical Psychology*. https://doi.org/10.1002/jclp.20240.

King, L. A. (2001). The health benefits of writing about life goals. *Personality and Social Psychology Bulletin, 27*(7), 798–807. https://doi.org/10.1177/0146167201277003.

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The "Trier Social Stress Test" - a tool for investigating psychobiological stress responses in a labor atory setting. *Neuropsychobioloy, 28*, 76–81. Retrieved from http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=Retrieve&list_uids=8255414&dopt=abstractplus%5Cnhttp://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=8255414PubMed.

Krause-Utz, A., Oei, N. Y. L., Niedtfeld, I., Bohus, M., Spinhoven, P., Schmahl, C., et al. (2012). Influence of emotional distraction on working memory performance in borderline personality disorder. *Psychological Medicine, 42*(10), 2181–2192. https://doi.org/10.1017/S0033291712000153.

Kuo, J. R., & Linehan, M. M. (2009). Disentangling emotion processes in borderline personality disorder: Physiological and self-reported assessment of biological vulnerability, baseline intensity, and reactivity to emotionally evocative stimuli. *Journal of Abnormal Psychology, 118*(3), 531–544. https://doi.org/10.1037/a0016392.

Kuppens, P., Oravecz, Z., & Tuerlinckx, F. (2010). Feelings change: Accounting for individual differences in the temporal dynamics of affect. *Journal of Personality and Social Psychology, 99*(6), 1042–1060. https://doi.org/10.1037/a0020962.

Linehan, M. M. (1993). Skills training manual for treating borderline personality disorder. 1993 (Vol. Diagnosis). Retrieved from http://www.loc.gov/catdir/bios/guilford051/93015216.html.

Lobbestael, J., & Arntz, A. (2015). Emotional hyperreactivity in response to childhood abuse by primary caregivers in patients with borderline personality disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 48*, 125–132. https://doi.org/10.1016/j.jbtep.2015.03.005.

Meevissen, Y. M. C., Peters, M. L., & Alberts, H. J. E. M. (2011). Become more optimistic by imagining a best possible self: Effects of a two week intervention. *Journal of Behavior Therapy and Experimental Psychiatry, 42*(3), 371–378. https://doi.org/10.1016/j.jbtep.2011.02.012.

Pfaltz, M. C., Schumacher, S., Wilhelm, F. H., Dammann, G., Seifritz, E., & Martin-Soelch, C. (2015). Acoustic emotional processing in patients with borderline personality disorder: Hyper- or hyporeactivity? *Journal of Personality Disorders, 29*(6), 809–827. https://doi.org/10.1521/pedi_2015_29_176.

Pfaltz, M. C., Wu, G. W. Y., Liu, G., Tankersley, A. P., Stilley, A. M., Plichta, M. M., et al. (2017). Cognitive and emotional processing of pleasant and unpleasant experiences in major depression: A matter of vantage point? *Journal of Behavior Therapy and Experimental Psychiatry, 54*, 254–262. https://doi.org/10.1016/j.jbtep.2016.09.001.

Posner, J., Russell, J. A., & Peterson, B. S. (2008). The circumplex model of affect: An integrative approach to affective neuroscience, cognitive development, and

psychopathology. *Development and Psychopathology, 17*(3), 715–734. https://doi.org/10.1017/S0954579405050340.
Prehn, K., Schulze, L., Rossmann, S., Berger, C., Vohs, K., Fleischer, M., et al. (2013). Effects of emotional stimuli on working memory processes in male criminal offenders with borderline and antisocial personality disorder. *World Journal of Biological Psychiatry, 14*(1), 71–78. https://doi.org/10.3109/15622975.2011.584906.
Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*(3), 385–401. https://doi.org/10.1177/014662167700100306.
Rosenthal, M. Z., Fang, C. M., & Chapman, A. L. (2015). Ambulatory measurement of emotional dysfunction in borderline personality disorder. *Current Opinion in Psychology, 3*, 75–79. https://doi.org/10.1016/j.copsyc.2015.02.008.
Rosenthal, M. Z., Neacsiu, A. D., Geiger, P. J., Fang, C., Ahn, R., & Larrauri, J. (2016). Emotional reactivity to personally-relevant and standardized sounds in borderline personality disorder. *Cognitive Therapy and Research, 40*(3), 314–327. https://doi.org/10.1007/s10608-015-9736-y.
Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology, 39*(6), 1161–1178. https://doi.org/10.1037/h0077714.
Rutten, B. P. F., Hammels, C., Geschwind, N., Menne-Lothmann, C., Pishva, E., Schruers, K., et al. (2013). Resilience in mental health: Linking psychological and neurobiological perspectives. *Acta Psychiatrica Scandinavica, 128*(1)https://doi.org/10.1111/acps.12095.
Santangelo, P., Reinhard, I., Mussgay, L., Steil, R., Sawitzki, G., Klein, C., et al. (2014). Specificity of affective instability in patients with borderline personality disorder compared to posttraumatic stress disorder, bulimia nervosa, and healthy controls. *Journal of Abnormal Psychology, 123*(1), 258–272. https://doi.org/10.1037/a0035619.
Schaefer, A., Nils, F., Sanchez, X., & Philippot, P. (2010). Assessing the effectiveness of a large database of emotion-eliciting films: A new tool for emotion researchers. *Cognition & Emotion, 24*(7), 1153–1172. https://doi.org/10.1080/02699930903274322.
Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods, 7*(2), 147–177. https://doi.org/10.1037/1082-989X.7.2.147.
Schroevers, M. J., Sanderman, R., Van Sonderen, E., & Ranchor, A. V. (2000). The evaluation of the Center for Epidemiologic Studies Depression (CES-D) scale: Depressed and Positive Affect in cancer patients and healthy reference subjects. *Quality of Life Research, 9*(9), 1015–1029. https://doi.org/10.1023/A:1016673003237.
Seebauer, L., Arthen, T., Austermann, M., Falck, J., Koch, L., Moulds, M. L., et al. (2016). Mood repair in healthy individuals: Both processing mode and imagery content matter. *Journal of Behavior Therapy and Experimental Psychiatry, 50*, 289–294. https://doi.org/10.1016/j.jbtep.2015.10.005.
Sewart, A. R., Zbozinek, T. D., Hammen, C., Zinbarg, R. E., Mineka, S., & Craske, M. G. (2019). Positive affect as a buffer between chronic stress and symptom severity of emotional disorders. *Clinical Psychological Science, 216770261983457*https://doi.org/10.1177/2167702619834576.
Sieswerda, S., Arntz, A., & Wolfis, M. (2005). Evaluations of emotional noninterpersonal situations by patients with borderline personality disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 36*(3), 209–225. https://doi.org/10.1016/j.jbtep.2005.05.004.
Staebler, K., Gebhard, R., Barnett, W., & Renneberg, B. (2009). Emotional responses in borderline personality disorder and depression: Assessment during an acute crisis and 8 months later. *Journal of Behavior Therapy and Experimental Psychiatry, 40*(1), 85–97. https://doi.org/10.1016/j.jbtep.2008.04.003.
Tugade, M. M., & Fredrickson, B. L. (2004). Resilient individuals use positive emotions to bounce back from negative emotional experiences. *Journal of Personality and Social Psychology, 86*(2), 320–333. https://doi.org/10.1037/0022-3514.86.2.320.
Vazquez, C. (2017). What does positivity add to psychopathology? An introduction to the special issue on 'positive emotions and cognitions in clinical psychology. *Cognitive Therapy and Research, 41*(3), 325–334. https://doi.org/10.1007/s10608-017-9847-8.
Verbeke, G., & Molenberghs, G. (2000). *Linear mixed models for longitudinal data.* New York: Springer.
Verschuere, B., & Tibboel, H. (2011). De Nederlandstalige versie van de McLean Screening Instrument for borderline personality disorder (MSI-BPD) [The Dutch version of the McLean Screening Instrument for borderline personality disorder (MSI-BPD)]. *Psychologie & Gezondheid, 39*(4), 243–248. https://doi.org/10.1007/s12483-011-0046-0.
Watson, D., & Clark, L. (1999). The PANAS-X manual for the positive and negative affect schedule-expanded form. *Iowa Research Online, 277*(6), 1–27. https://doi.org/10.1111/j.1742-4658.2010.07754.x.
Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063–1070. https://doi.org/10.1037/0022-3514.54.6.1063.
Wichers, M., Peeters, F., Geschwind, N., Jacobs, N., Simons, C. J. P., Derom, C., et al. (2010). Unveiling patterns of affective responses in daily life may improve outcome prediction in depression: A momentary assessment study. *Journal of Affective Disorders, 124*(1–2), 191–195. https://doi.org/10.1016/j.jad.2009.11.010.
Zanarini, M. C., Vujanovic, A. A., Parachini, E. A., Boulanger, J. L., Frankenburg, F. R., & Hennen, J. (2003). A screening measure for BPD: The McLean screening instrument for borderline personality disorder (MSI-BPD). *Journal of Personality Disorders, 17*(6), 568–573. https://doi.org/10.1521/pedi.17.6.568.25355.